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ACUTE RHEUMATISM.

by

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ACUTE RHEUMATISM.

The name "rheumatism" was given to the disease as the pain, which is such a prominent symptom, was thought to be due to the "rheum" (Greek *ῥέω* I flow) flowing down from the brain and lodging in the painful parts.

Earlier writers confused what we now distinguish as Acute Rheumatism or Rheumatic Fever with gout, rheumatoid arthritis and other diseases with joint troubles such as pyaemia and gonorrhoea. To Sydenham we owe the first full and accurate description of the clinical aspect of the disease. As we must now, from the researches of Poynton, Paine, Beattie, Shaw, Ainley Walker and others regard its bacterial origin as proved we can describe Rheumatic Fever as a general acute infection, caused by the presence of the micrococcus rheumaticus, characterised by fever, peculiar acid sour smelling sweats, painful swellings of the joints, and by the affection in various degrees of the heart in its peri, endo and myocardium.

AGE INCIDENCE: Rheumatic Fever occurs at all ages (except in babies under one year). The greatest incidence is in the second decade of life. After thirty it is comparatively uncommon, and it has also

been found that the liability to heart damage is less among those who have their first attack after that age. One attack predisposes to another and each attack leaves the heart more vulnerable. In children the disease is not characterised by the same degree of joint swelling as in adults, nor are the acid sweats so marked, but it is none the less serious as in them the heart is almost invariably affected by endo- myo- or pericarditis leading to permanent affections of the valves when recovery takes place. That the heart seldom escapes is proved by the fact that (in 250 fatal cases in children) in only three was the mitral valve found to be quite healthy.

MORTALITY: Referring to the Registrar General's returns for 1910 we find that 1998 deaths occurred in England from "Rheumatic Fever". For London in 1913 the figure given is 220. These figures must not be taken as giving the real number of persons who die annually of the consequences of Acute Rheumatism as most of the deaths returned as due to "heart disease" are actually deaths from valvular lesions left by rheumatic endocarditis. The fallacy is well shown by contrasting the figures I have just given with the following:- In 1913 6658 deaths occurred in London from pericarditis, endocarditis and organic heart disease. In 1911 we have 18,258 deaths re-

corded for all England from non-infective endocarditis (practically rheumatic endocarditis). One has only to go round the medical wards of any hospital and enquire into the history of the many heart cases one finds there, to be fully convinced that Rheumatic Fever is a most potent factor in filling those wards and eventually swelling the death returns of this country.

AETIOLOGY: Various theories as to its causation have at various times been propounded. In view of our present greater knowledge it will be sufficient to mention the chemical theory of Prout; and that of J. K. Mitchell, ~~Can~~stall, Day and others that it was in some way caused by nervous disorders.

Most authorities seem now to accept it as proved that a micro-organism is the cause. This we will consider in fuller detail later.

Seasonal and climatic conditions undoubtedly play a part. Acute Rheumatism as one would expect, is most common in autumn. Exposure to cold, especially damp cold, certainly often causes the illness in those predisposed by heredity or previous attack. Fatigue and mental worry are also factors in the production of attacks.

It is met with all over the world, but I have been told by medical men practising in the tropics that they seldom see it. One man in seventeen years

practice in Singapore had never had a case of Acute Rheumatism although chronic rheumatism is common and most unamenable to treatment.

It is frequently met with in countries where there is much difference between the day and night temperatures (Church). Dr. Burton Brown's experience seems to throw doubt upon this statement. In India Acute Rheumatism was very seldom seen which was remarkable seeing what sudden changes of temperature were experienced there. His experience seemed to disprove the idea that rheumatism was due to chill. (Burton Brown, R.S.M. Vol.11, pt.111, p.43./.) Having practised both in the North of England (Gateshead-on-Tyne) and in the South (Kent) I have been much struck by the greater frequency with which one met with it in the former district. The men in Kent, mostly employed in farm work out of doors and therefore frequently at work all day in wet clothes, suffered much less than those in Gateshead mainly employed in factories and so under cover. This seems to support the idea of its being an infection. Dr. Newsholme in 1895 supported the infective theory with statistical evidence which seemed to bear out his contention that Acute Rheumatism should be reckoned among the infectious diseases. In this connection one might mention that it has been observed that frequently several cases occur at different times in the same

house. One observer states that he had attended twelve cases in the same house in three years. Some years ago a run of cases of pneumonia occurred in a training ship at Queensferry and of course we now know that pneumonia is a germ disease.

Evidence in support of the bacterial causation of Acute Rheumatism:

More than twenty years ago Mantle (B.M.J. 1887, Vol.I, p.1381) drew attention to throat infection in cases of Acute Rheumatism and isolated a diplococcus (similar to that which he had found in the tonsils) from the fluid effused into the joints of the patient. In 1891 Achalme discovered an anaerobic anthrax-like bacillus which he considered to be a causal organism. (Compt. rend Soc. de Biol. Paris 1891). Poynton and Paine were unable to find this bacillus, but in 1899 discovered a diplococcus in eight cases where the diagnosis of Acute Rheumatism had been confirmed post mortem. They regarded it as identical with the organism described by Triboulet in 1897 and by Wassermann in 1899. This diplococcus was found in the most congested parts of the synovial membrane, in the deeper parts of the visceral pericardium, in the fibrino-cellular exudation, in the parietal pericardium, in the rheumatic nodules and in the kidney. They were also successful in isolating it from the throat of a man suffering from Acute Rheumatism and acute faucial catarrh. (Lancet 1900 Vol.II, pp.860 & 962).

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Cultivations of this organism inoculated into rabbits intravenously produced vegetations on the valves, pericarditis and arthritis.

The rabbits during life showed swelling of the joints of the legs, and that these swellings were painful was obvious from their holding up their feet and limping about. It was noticed that exposure to cold aggravated the symptoms, which were relieved almost at once when the animals were placed in a warmed room.

Suppuration was never produced when this diplococcus was injected alone. Very different were the effects of injection of staphylococcus aureus. It produced either septicaemia and rapid death with marked haemolysis but no local lesion, or in other cases pyaemia with abscess formation in the kidneys, spleen and heart wall.

The investigation of the effects of inoculation of the latter organism was undertaken to disprove Singer's statement that Rheumatic Fever was an attenuated form of pyaemia: he had been led to make this assertion by having found staphylococcus pyogenes aureus in the urine of seventeen cases of Acute Rheumatism. (Wiener Klin. Wchnschw. 1894, No. 26, p. 449)

In 1899 also Malkoff in a communication "On the infectious character of Rheumatic Fever and the connection between it and Chorea" had described a diplococcus, found in a fatal case of Acute

Rheumatism and Chorea, which injected into rabbits produced fever and multiple arthritis. The micro-organism was found in the fluid in the affected joints of the rabbits: cultures reproduced the disease (Berl. Klin. Wchnschw 1899, No.29, p.638).

Poynton and Shaw have recorded in their account of their experiments with diplococcus rheumaticus and staphylococcus aureus that, when the staphylococcus was injected, if the rabbit reacted at all, death always followed with the symptoms and post mortem appearances of septicaemia or pyaemia (described above p.6) With the diplococcus "recovery is not uncommon and this after very definite signs of infection have developed; or again chronic articular inflammation, with osteo-arthritic changes may result and persist for months."

It thus appears that the produced diseases resemble in the one case septicaemia and pyaemia, and in the other Rheumatic Fever as we are accustomed to see these diseases clinically. (Trans. Path. Socy. London Vol. LV, p.134.)

Beattie (Journal Pathology and Bacteriology, Vol.XIV, p.432), confirms Poynton and Paine's results. He has repeatedly been able with pure cultures of the diplococcus to produce polyarthritis, synovitis, valvulitis and pericarditis without suppurative change (so distinguishing it from the ordinary streptococci). The arthritis presented the peculiar clinical

features of the arthritis of acute rheumatism. He says "The arthritis produced presents the main features of that seen in acute or subacute rheumatism in the human subject, viz. the rapidity and even suddenness with which the inflammation flies from one joint to another; the subsidence in one joint with an increased intensity of reaction in another; the tendency to relapses without obvious cause and also as a result of exposure to cold; and the absence of any gross anatomical changes in the joints."

W. V. Shaw conducted a series of experiments on monkeys and rabbits with a strain of the diplococcus obtained from Beaton and Walker (to whose results I refer next). He concludes that, as this organism produced the majority of the lesions clinically associated with acute rheumatism (arthritis, pericarditis, myocarditis and endocarditis) it followed that it was "the actual infective and causal agent of acute rheumatism" (Journal Path. and Bact. Vol.IX, p.171).

Beaton and Ainley Walker, B.M.J. 1903, Vol.I, p.237 reported as follows:- "We are now able to state that we agree entirely with the assertion that the micrococcus is constantly associated with acute rheumatic lesions and is the causal agent in their production. We believe the coccus which we have isolated from rheumatic cases to be identical with

that obtained by Triboulet and Wassermann, Poynton and Paine and others." They add that the same organism can be obtained from cases of chorea and on injection into animals may produce a typical attack of acute rheumatism just as do cultures from cases of the latter.

The various observers, whose conclusions are given above, are fairly well agreed as to the appearance and character of the organism and as to the conditions under which it can be cultivated.

Poynton and Paine describe it as a diplococcus; Beaton and Ainley Walker as a tiny micrococcus arranged in pairs and short chains in films from ordinary sub-cultures. It is very small ($.5\mu$ in diameter) and is difficult to demonstrate in human tissues but is more easily shown in animals (killed in the height of the disease). When growing in the human tissues the majority of the cocci are seen in pairs; on culture media it is difficult to distinguish it from an ordinary streptococcus, but the experiments on animals differentiate it from the others.

The micrococcus grew best anaerobically in a slightly acid medium. In the various media it produced formic acid in considerable quantity. Later on we shall notice that formic acid is found in the urine in Rheumatic Fever.

It retains the stain by Gram and in the Weigert modification of this method. It stains well with

all ordinary dyes; is not capsulated: is not agglomerated either by the serum of convalescent rabbits or of man.

Summary: It can be cultivated on artificial media outside the living body; on inoculation into animals it gives rise to the characteristic lesions of the disease and can be isolated from these lesions; it therefore fulfils Koch's conditions.

It is only fair to add that many eminent physicians are still of opinion that this "micrococcus rheumaticus" (Beaton and Walker) may not be the cause of Acute Rheumatism. Osler (System of Medicine, p.373) regards the matter as still "under discussion" as do Muir and Ritchie. The latter sum up as follows.

"The facts already accumulated speak strongly in favour of this organism being causally related to Rheumatic Fever though this cannot be considered completely proved." (Muir and Ritchie, p.229).

Guthrie (Royal Society of Medicine, Vol.I, pt.3, Sect.Therapeut.p.66) concludes that: "The specific nature of the organism is still sub judice."

Stockman also speaks "of the present indeterminate state of our knowledge regarding the specific organism of Rheumatic Fever." (R.S.M. Vol.II, pt.3, Sect.Therapeut. p.33). Many investigators have been unable to find the coccus but in this connection Lees remarks that one positive observation outweighs many negative ones which might be due merely to slight differences in the precise method adopted. He draws attention to two pieces of evidence.

What is it?

(1) A rheumatic nodule of recent origin in a child in St.Mary's Hospital, when excised by Dr. Poynton under aseptic precautions, at once placed in a nutritive medium and incubated for 48 hours showed on section an exuberant growth of diplococcus in pure culture.

(2) Dr. Willcox states that a diplococcus had been obtained from the blood of more than one of his rheumatic patients during life and that in one of these cases the infection was certainly not "terminal" for the patient is still alive and in good health 8 yrs. after the venesection. (R.S.M.Vol.II, pt.3, Sec.Ther.p.48)

Whether we regard it as proved or not that the micrococcus rheumaticus is the cause, it will be generally admitted that certain persons are peculiarly predisposed to rheumatism, that this predisposition is hereditary and is favoured by residence in cold damp localities and by certain occupations.

MORBID ANATOMY: Death during the attack is rare and in the few post mortems that have been obtained it cannot be said that any pathological change characteristic of Rheumatic Fever has been observed. The affected joints contain a sero-fibrinous fluid showing excess of leucocytes under the microscope: pus is practically never found. The synovial membrane is thickened and boggy. In cases where death has

resulted from hyperpyrexia the appearances are simply those of death from fever; in two cases however definite evidence of meningitis was observed. The usual evidences of pericarditis, endocarditis and myocarditis are found in cases where the rheumatic process has fatally attacked the heart. If death has ensued in a first attack there may be effusion into the pericardial sac, lymph adhering to both pericardial surfaces, some degeneration of the heart muscle, dilatation of the cavities but invariably, vide ante (p. 2) affection of the valves (especially on the left side of the heart). There is sometimes merely roughening of the surface, in other cases vegetations of varying size, which may have discharged their centres and left ulcers. The micro-organism (as we have already seen) can be demonstrated in the deeper layers of the valve substance surrounded by new cells: the surface of the valve showing the inflammation resulting from its action. Should it be a case where there has been one or more previous attacks of pericarditis, the pericardium will probably be found thickened both in its visceral and parietal layers and will show fresh layers of lymph on both surfaces: when these surfaces are separated a "honey-comb" appearance suggestive of tripe is presented. The pericardial sac may be obliterated. If the endocarditis is old standing the ulcers will now be healed

and the valve segments shrunk. Different stages of the process in the valves may be seen in the same case. Poynton has investigated the structure of the subcutaneous nodules so frequently seen in fatal cases of Acute Rheumatism in children and finds them composed of a central core of fibrin surrounded by concentric layers of proliferating fibrous tissue and nucleated spindle shaped and round cells. There may be softening of the liver, spleen and kidneys but they may be quite normal.

SYMPTOMS: As the symptoms and onset differ greatly in adults and children it will be convenient to describe firstly the adult type and secondly the disease as we see it in the young.

Adults: The onset is usually fairly sudden. After some exposure the patient complains of a chilly feeling, more or less sore throat and pains in one or more joints. The temperature speedily rises to 102°, 103° or even higher and continues up with irregular remissions for varying periods. In slighter cases it falls to normal at the end of a week but may not do so for three or four weeks or even longer. The evening temperature often continues to rise to 99 or 99.6 or so for some time after the morning temperature has become normal. While the fever lasts the patient is drenched in sour acid swelling sweats.

The odour is most characteristic and is a valuable help in diagnosis. Even though the patient be frequently sponged and his clothing changed several times a day the smell does not go.

The joints affected are usually the knee, wrist, ankle and those of the fingers. The joints are swelled and very painful as I know from experience. Movement of them is impossible without causing great pain and a jar of the bed is torture. The swelling and pain fly from one joint to another. The joint which has apparently recovered one day is often swelled and as painful as ever on the following day. The skin over the affected joints has a reddish colour but not the brawny shining appearance of a gouty big toe. The tongue is covered with a thick coating of white fur ("the rheumatic blanket") and in most cases of Acute Rheumatism there is "sore throat". The pharynx, uvula and tonsils are inflamed and congested so that considerable pain and difficulty in swallowing are experienced. Robert Hutchison considers that rheumatic tonsillitis particularly involves the tendons of the muscles (the tensor palati and the levator palati) and so produces pain in upward and downward movement. "Diseases of Children", p.165.

Many cases commence with sore throat and fever and only develop the joint affections some days later. I have observed that in cases of Rheumatic Fever there

is often a history of repeated attacks of tonsillitis. As we have seen already the diplococcus rheumaticus has been found in the tonsils and by many it is believed that it is through the tonsils that the organism gains access and sets up Acute Rheumatism. In addition we have the usual symptoms of a case of fever. Headache (often intense), loss of appetite, constipation (although I have seen cases ushered in with diarrhoea and sickness). The urine is scanty and high coloured; it deposits urates and may contain a trace of albumin. Lately it has been stated that formic acid is constantly present in considerable quantities. The thirst is so intense that the patient usually drinks very large quantities of fluid and yet the quantity of urine passed is always less than normal. Sweat rashes are naturally common. Small vesicles appear filled with clear fluid which later on becomes thick and sometimes purulent. Various other skin affections have been observed such as erythema multiforme and erythema marginatum and nodosum. In a few cases purpuric rashes have occurred. In several cases I have observed a general peeling of the skin during convalescence resembling that of scarlatina.

There is not much tendency to delirium although the patient is generally sleepless owing to his sore throat and his joint pains.

Pulse rate is increased but it is not as fast as one would expect from the temperature and it is not usually irregular in the absence of heart complications.

COMPLICATIONS: Endocarditis is so common that it should hardly be described as a complication but rather as a part of his illness which the rheumatic fever patient is sometimes lucky enough to escape. As we have seen the older the patient the more likely is his heart not to suffer provided that sufficient rest (after the actual fever goes) be given. Stephen Mackenzie states that of 116 cases 58.1 per cent developed endocarditis in the first attack, 63 per cent in the second and 71 per cent in the third (Osler) (p. 375). Osler gives 35% as his experience. Taylor says that between one third and one half of his cases were afterwards found to be suffering from valvular lesions (System of Medicine). The onset of endocarditis may be shown by an increase of rate and irregularity of the pulse with rise of temperature, usually accompanied by the development of a soft blowing murmur in the mitral area. Again no signs may be observed during the illness and yet the patient later on may develop typical signs of valve mischief. To hear a murmur does not mean that the patient is bound to have valve disease as such may be well heard during the illness and disappear completely during convalescence. These murmurs are probably due to

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dilatation of the cavity of the heart and stretching of the valves or may be haemic as anaemia quickly develops (see later).

Pericarditis occurs in 10 per cent. Poynton states that in 250 fatal cases under 12 years of age the pericardium was affected in 215. That is 86%.
Practit. Oct. 1914.
It is very much more common in children. It may occur at any stage of the pyrexia while endocarditis is more common at the beginning. There may be no symptoms, merely the physical signs of friction (to-and-fro rub). If there is effusion there will be increased cardiac dulness which may extend an inch outside the nipple line and an inch or more to the right of the middle line. In such cases there will be increased frequency and irregularity of the pulse, pain and oppression over the praecordia. These symptoms may be present without any effusion. The effusion consists of large flakes of lymph and serum. Purulent effusion is not common in a pure case. Pericarditis is a serious complication but recovery, even when the amount of fluid is considerable, is not uncommon. There is a difference of opinion as to whether pericarditis ever occurs without endocarditis. There is general agreement that in pericarditis it is the myocardial damage which is the great element in the immediate or subsequent heart failure but it is the pericarditis which enables us in many cases to

detect the grave nature of the attack. The pericardial inflammation sometimes extends to the pleura but pleurisy and pneumonia (more rarely) sometimes occur without any signs of pericarditis.

Myocarditis leading to fatty degeneration of the heart muscle and dilatation of the heart is probably present in all cases where pericarditis or endocarditis have occurred.

Fisher states that myocarditis may occur without any signs of either of the other more easily diagnosed cardiac complications. (Bristol Med. Chir. Journ. Vol. XVIII, p. 21).

Nervous complications: Under this heading we may include hyperpyrexia, a condition in which the temperature without any obvious cause suddenly goes up to 106° , 107° or even 110° . Fortunately it is not a common complication as the mortality is high. It may come on without warning but usually the joint pains suddenly become less while the patient becomes more restless with a hot dry skin and an increasing pulse rate; as the temperature goes up the restlessness becomes delirium; later on stupor supervenes passing into coma. Hyperpyrexia has been most frequently observed in first attacks and in men. Death may take place within eighteen or twenty-four hours of the time the temperature began to rise.

Other Nervous Complications: Occasionally meningitis occurs and multiple neuritis has been noticed. Chorea in adults is rare. Insanity with suicidal and homicidal tendencies I have myself observed in a man of 35. Delusions remained after several days of delirium (not due to overdose of salicylate). Eventually he had to be certified but recovered after six months detention in an asylum.

Anaemia occurs early in the disease and is as noticeable clinically as it is demonstrable microscopically. Malassez found that the red blood corpuscles may fall to two millions in the cubic millimetre. (Dict. Encyclop. des. Sc. mèd article Rheumatism, p. 492)

Hayem states "Acute articular rheumatism is one of the diseases most destructive to the corpuscles.... in a really acute case, even though not a long one, the diminution is rarely less than a million.

The variations in the worth of the corpuscles depend usually upon the form of the crisis." (Du Sang '89, p. 916)

Garrod subsequently investigated this question of blood destruction; he agrees with Hayem's conclusions but did not find that the depreciation in value of the corpuscles was progressive as Hayem had thought. Garrod says, "An attack of rheumatism is always attended with a considerable diminution in the number of red blood corpuscles. The fall commences early in the attack is usually extremely rapid, in some cases a million in four or five days." (Med. Chir. Trans. Vol. LXXV, p. 192)

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He adds that the blood curve affords a far more delicate guide to the activity of the rheumatic process than the temperature curve gives us.

Leucocytosis: There is a moderate degree of leucocytosis during the febrile stage: as the patient improves the number of leucocytes falls, to go up at once if a relapse occurs.

The acute oligocythaemia is one form of the anaemia of acute rheumatism: the other is a pseudo-chlorotic condition which occurs later and if not treated, may last for a long period although in other respects the patient may be much better. In fact this anaemia is apt unfortunately to persist for weeks or months. It causes shortness of breath and murmurs in the heart which may lead one to suppose that the heart has sustained permanent damage. It aggravates the symptoms of organic heart mischief and retards the recovery of the myocardium which, specially during convalescence, requires to be nourished with blood containing plenty of iron.

Acute Rheumatism in childhood presents a totally different clinical picture. There is seldom as much fever; in many cases hardly more than a degree. The profuse sweats are wanting. Joint swellings and pains are much less in evidence and may in fact be quite absent. The signs of illness may indeed be so slight that children suffering from rheumatism are often sent to school while active endocardial

mischievous (which is only recognised too late) may be going on. Rheumatic fever in children is "an affair of the heart". Endocarditis and pericarditis being much more common in them than in adults. Lees says "We must insist on the conception that it is a microbic toxæmia most virulent in early life in which the heart is invariably affected to a greater or less degree, but the joints slightly or not at all." (R.S.M. Vol.II, part 3, Sect.Ther.p.45) See ante "Age Incidence", p.1.

Chorea: is a common attendant on Acute Rheumatism in childhood. It may precede the rheumatic attack, may occur during it, or may follow later. The discovery of the micrococcus in the ventricular fluid is interesting as chorea has by many been regarded as cerebral rheumatism.

Subcutaneous Nodules: Although very occasionally noticed in adults, it is in children that these peculiar little growths under the skin are specially found, and more particularly in association with pericarditis and endocarditis. They are regarded as of evil omen but there seems to be no doubt that cases in which they have been observed do recover with no valve damage (Whitla). The nodules may be so small as to be difficult to find but may attain the size of a small haricot bean. They grow under the skin (which is freely moveable over them) in the fibrous tissue, usually in the neighbourhood of the

knee and elbow joints, on the tendons of the wrist, on the scalp (along the occipital ridge), over the vertebral spines and over the scapula.

Their structure I have described under post mortem appearances (p. 13).

DIAGNOSIS: I. In adults there ought to be no difficulty in recognizing a typical case. Pyaemia and gonorrhoeal "rheumatism" both present joint affections with pyrexia but in the former the swelling and pains do not fly from joint to joint (with rapid recovery in the first joint) as in Acute Rheumatism: the acid sweats are replaced by a hot burning skin and there may be some obvious source of the pyrexia. Rigors are common in pyaemia. In Acute Rheumatism there is usually only some shivering at the beginning; in the latter we have the history or presence of urethral discharge and the local features (pain and swelling of one joint usually) are more pronounced than the general (fever etc.) In any case sodium salicylate will soon decide between them.

Arthritis deformans beginning acutely can hardly be distinguished from Rheumatic Fever in the early days of the illness. Later it will be noticed that there is more thickening round the joints than one sees in Acute Rheumatism where it is quite exceptional to find any persisting alteration in the appearance of the joint.

In malarial countries Acute Rheumatism may be mistaken for malarial fever. This has occurred within the writer's experience. The persistence and character of the fever and especially the failure of quinine to influence its course should be sufficient to show that it is not malaria.

Glanders has been diagnosed as Acute Rheumatism but this mistake could only be possible in its early stages.

II. Children: We have already seen that in children Acute Rheumatism is not usually an arthritis but in the rarer cases in which there is joint affection with some rise of temperature the diagnosis will be fairly easy. Commonly the only signs will be slight fever and evidences of endocarditis or possibly pericarditis. We have discussed the physical signs of both conditions. In addition one might mention that a reduplication of the second sound at the apex may be the only sign of heart trouble. Frequently the diagnosis is only made much later when the acute process is past.

Acute osteomyelitis must not be diagnosed as rheumatism. The tense swollen appearance of the skin over the acutely inflamed bone and the tenderness on deep pressure are points to be noted as is the much higher temperature, hot dry skin and the look of acute illness. In cases where doubt still

exists operation will clear up the diagnosis with little harm to the rheumatic patient and great benefit to the subject of acute necrosis.

Tonsillitis: It is often difficult to draw the line between rheumatic tonsillitis and rheumatic fever. This difficulty is best got over by regarding all cases of tonsillitis (except that of diphtheria and the like) as examples of acute or sub-acute rheumatism and treating as such.

The so-called "growing pains" of childhood are evidences of rheumatism and not of a physiological process as was so commonly believed until quite recently, by the public, at any rate.

Infantile Scurvy is sometimes mistaken for rheumatism owing to the tenderness in the limbs. The age here will help us to distinguish between them. Acute Rheumatism does not occur in very young children and never in those under one year of age.

PROGNOSIS: In a simple case of acute rheumatism without serious heart complications is good as regards recovery. At the commencement of the attack the greatest caution must be exercised in giving a prognosis. A full recovery to former health and vigour can only be looked for in cases where the heart has escaped serious damage. Under what circumstances this is most likely has already been discussed under "Age Incidence."

Children who are well cared for are naturally more likely to escape other attacks than those less happily circumstanced.

In cases where there have been cardiac complications Cantley (Amer. Med. 1913 I, p.348) considers that the outlook depends on the state of the heart muscle: he points out that obstruction is harder to compensate for than insufficiency. Much hypertrophy or dilatation are of bad omen. The loudness of the bruit is no measure of its serious significance. "In rheumatic cases much depends on the degree to which the child proves prone to active outbursts of the infection; this depends in part on heredity, in part on social and climatic environment."

TREATMENT: At the first sign of any illness which suggests acute rheumatism the patient should be put to bed. He should be clad in flannel and placed between blankets. The mattress should be a hair one on a spring bed. A fire is advisable except in very warm weather. The window should be open, and widely so, except on a wet day as the atmosphere quickly becomes unpleasant from the peculiar odour given off by the patient's body. The temperature of the room should be about 65° F. If the bowels have not been freely opened a dose of calomel followed by a saline should be given. The diet should be milk; half a pint every two hours, but

it is advisable not to drink the whole half pint at once as it then forms one large curd in the stomach. Thirst may be alleviated by barley water flavoured with lemon. Aerated lime water or soda water may be given with the milk.

Drugs: Large doses of sod. salicylate should be administered at once; gr. X every two hours or gr. XV every three hours. (In cases with very high temperature gr. X may be given hourly until there is a response). In each case we give a double quantity of bicarbonate of soda with the salicylate. These large doses should be continued until the temperature goes down nearly to normal or until the patient complains of deafness and buzzing in his head or shows signs of mental disturbance (delirium). If these appear, the dose is reduced but must be increased again as soon as possible unless the temperature should have fallen to normal in the meantime. If he shows any alarming symptoms it may be necessary to stop the salicylate altogether for 24 or 36 hours but there is no danger and not much risk of any alarming symptoms so long as the bowels are kept freely open. In supposed salicylic acid poisoning the most reliable test is the presence of acetone in the urine. (W. H. Willcox, R.S.M., Vol.II, Thera.Sec.) part III/. This can be determined by a chemical test. Lees (R.S.M. Vol.II, part III, p.38/ has Thera. Sec.)

emphasized the value of the addition of the bicarbonate of soda as a means of diminishing the unpleasant effects of large doses of the salicylate. He considers that the bicarbonate of soda also helps to neutralise the acid toxins of the microbe which have been found to be partly formic and partly acetic acid.

Sodium bicarbonate should in any case be given in sufficient quantity to render the urine slightly alkaline.

The treatment by drugs of the salicylic acid group (which includes besides the acid and its soda salt, salicin, aspirin, nova-aspirin, salophen etc.) has practically displaced the alkaline treatment formerly in vogue, but some eminent physicians still prefer the latter. In it potass acetat or potass citrat are given every three hours in doses of gr.15 of each until the urine is alkaline & thereafter in large enough doses to keep it so. Roberts in his new edition still advocates this treatment. Clifford Allbutt (last edition) states that the alkaline treatment by depressing the heart's action tends to lessen the danger of the occurrence of pericarditis and possibly of endocarditis. There is a general concensus of opinion that the salicylates relieve the pain and reduce the temperature. To this extent their action is as specific in Acute

Rheumatism as that of quinine in malaria. Unfortunately there is not the same agreement as to whether they lessen the risk of heart complications or the number of relapses. If, as many believe, salicylic acid has a specific action on the diplococcus it ought to hinder the development of endo and pericarditis and favourably affect their course. Certainly the lessening of the period of pyrexia must help the heart if only by checking the degeneration of the heart muscle (which we know occurs in all pyrexias) and the consequent dilatation of the organ. In this connection Poynton (B.M.J. 1913, Vol.II, p.785) says that in his experience large doses of salicylate are not well borne by delicate children and he is not persuaded that strong ones show any decisive proof that these drugs are specific.

Local applications if they do not reduce the swelling of the joint most certainly relieve the pain. I have found that hot soda fomentations give most relief (flannels wrung out of hot soda solution are applied to the joint, covered with a piece of white jaconet, large enough to cover it all in and secured by pieces of tape or bandage above and below the joint). Fuller's solution may also be used for fomentations: it consists of the following R_x Sod. bicarb } vi: tinct opii } $\frac{1}{2}$ glycerine } π ag ad $\frac{3}{4}$ X. Methyl salicylate may be made into a thin ointment

and rubbed gently into the joint and then covered with cotton wool or lint. Personally I believe that the warmth of the fomentation is what gives relief.

American physicians have recommended that the joints should be placed in splints or even in plaster of Paris.

Pericarditis: If signs of pericarditis appear the salicylates should not be stopped too quickly unless there is distinct evidence of weakening of the heart's action. Ammonium salicylate or salicin may be given in place of the soda salt. Especially if there is much pain and restlessness there is no drug so useful as opium given in the form of Tinct. opii. or nepenthe. Heroin may be tried. It is safer than morphia. As the pericarditic patient is usually a child all the opium group must be given with care. See foot-note p.34. If the pulse is becoming faster weaker and irregular, digitalis or strophanthus should be given, combined with liq. strych. hydrochlor; and the salicylate should be discontinued. Alcohol may also be usefully given in such cases. In an ordinary case of Acute Rheumatism it does harm in the acute stage.

Locally: Blisters, leeches or icebags may be applied. The icebag certainly seems to relieve the pain and may help to check the inflammatory process.

The patient's feet must be kept warm with hot bottles while the icebag is applied. If removal of the effused fluid is decided upon on account of its quantity causing great embarrassment of breathing or because it is proved to be purulent, on no account should it be done by aspiration in which there is every chance of the heart being pierced. Open operation is much to be preferred, a piece of the fifth costal cartilage being removed and the pericardium freely exposed before being incised. If aspiration is done the fourth or fifth interspace should be chosen. Removal of the fluid is only very rarely necessary; "pericardial effusion demanding paracentesis is an almost unknown complication of the disease," British Medical Association Meeting, 1913 (Heart Section).

Endocarditis: Most of what has been written above will apply to the treatment of endocarditis.

It is generally agreed that with due care salicylates may be continued. The same local applications as in pericarditis have each their own advocates. A small blister over the affected valve may be applied or one or two leeches may be employed to draw off a little blood in a vigorous adult but not in a child. The patient must be kept absolutely quiet and an icebag applied over the praecordium. So long as the pulse continues good no special heart treatment is

indicated but if it becomes quick small and irregular digitalis or strophanthus should be given in doses suitable to the age. Strychnine may be given with either or hypodermically. Brandy is useful: (good whisky is better than cheap brandy). Ether and ammonia may be given. Oxygen inhalations relieve the distress of the patient. If patient is very restless small doses of morphia may be given hypodermically. Potassium iodide is given by some, during convalescence with the idea of aiding absorption of vegetations etc. Iron, cod liver oil and malt are excellent during that time.

All cases require months of rest. (See later, p.33).

Hyperpyrexia: Here treatment must be prompt if it is to be of any use. Drugs are too slow in their action. We consider that the patient is threatened with hyperpyrexia if his temperature rises above 105° . Without any more delay than is involved in making preparations, the patient is placed in a bath at a temperature of 80° . Lumps of ice are added till the bath thermometer registers 60° . He may be kept in the bath for twenty minutes or until his temperature has fallen to 100° . He is then quickly dried, clad in warm clean pyjamas and got back to bed where he is surrounded with hot water bottles and carefully watched lest it may be necessary

to repeat the bath which should be done if his temperature goes up again to 104° . To give a bath to a man who is possibly delirious or unconscious requires at least two strong men or women. If sufficient help is not available we must fall back upon ice packs. The patient is wrapped in a sheet wrung out of iced water; pieces of ice are placed all round him and renewed as they melt or the surface of the body may be rubbed with lumps of ice.

CONVALESCENCE GENERALLY: Whenever the temperature falls soups, bovril, milk puddings, soufflés etc. should be given. Later on fish and chicken. Mutton should be given before beef but neither for several weeks after convalescence begins and then only in small quantities.

It is well to continue the salicylate for ten days after the temperature has become quite normal, say gr. X tds. with it give nux vomica and infus. gentian co. Later on when the tongue is quite clean iron, which is always urgently needed, should be given in an easily assimilable form such as Pil. Blaud gr. X, liq. arsenicalis $\mu 3$ in tablet form. *L. d. s* Fer Robin is a useful preparation which is tolerated by those who cannot take iron in the more common forms. The administration of iron should be continued until examination of the blood shows that the anaemia is cured.

The patient ought to be kept in bed for at least two weeks after the evening temperature becomes normal even in a mild case. It is most important that the temperature should be taken in the evening (and if need be the thermometer may be left with the patient so that this may be done).

In all cases where there have been cardiac complications two months rest in bed after the patient has got round the corner is none too long. Children ought not to be sent back to school for months after a serious attack.

I have not referred to the question of serum treatment as the results so far seem rather disappointing. It will suffice to mention that Stengel has stated that he noted improvement from the use of anti-streptococcic serum in three protracted cases.

Meuzer also claims to have successfully treated a number of cases with a serum made from streptococci of human origin. He gave daily injections of 5-10 c.c. till 50 c.c. had been given allowing in some cases where the fever was very high intervals of one or two days. He contends that there is less risk of endocarditis developing and that relapses and chronic conditions are not so frequent. So far others have failed to get the same benefits from this treatment. Poynton (B.M.J. 1913, Vol.II, p.785) looks with dismay at the giving of large doses

of streptococcal vaccines in acute carditis: he has seen pericarditis follow immediately on their use and has also seen mischief lighted up in cases where it was apparently quiescent.

Although there is a well demonstrated connection between tonsillitis and acute rheumatism it does not follow that removal of tonsils will prevent the development of rheumatism or quite obviate a relapse. The results have been disappointing in these respects but still all enlarged tonsils in children (rheumatic or otherwise) should be removed.

The treatment of rheumatic fever in view of the enormous number of damaged hearts one sees in practice cannot be said to be peculiarly successful, so in this disease as in many others the best treatment is Prevention by taking care that children are warmly clad and well shod in cold damp weather.

Footnote:

In very young children the does should be very small at first and increased gradually, the effect being carefully watched. The pupils are a good guide. Where there is much constitutional disturbance and cardiac disease the pupil is frequently somewhat dilated and the reduction of the size of the pupil requires larger doses.

C O N C L U S I O N S.

- I. We may now regard it as practically proven that Acute Rheumatism is a germ disease and that the microbe is the "micrococcus rheumaticus."
 - II. Acute Rheumatism is very frequently met with in children in whom it presents the symptoms of a carditis and does not usually exhibit the joint manifestations seen in older people.
 - III. Drugs of the salicylic acid group have an undoubted effect in relieving pain and reducing fever in this disease. Large doses can be profitably and safely given.
 - IV. Prolonged rest in bed is necessary in every case as, in all cases, the heart is more or less damaged; with such rest many cases escape permanent heart injury.
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